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ORIGINAL ARTICLES.

QUEER BEHAVIOR OF A BIT OF GLASS WITHIN THE EYE.

By CHAS. H. BEARD, M.D.,

CHICAGO.

G. W. G., aged thirty-two, a chemist for the Standard Oil Company, consulted me on March 10th, 1899, relative to an injury of the left eye. Two days before he had been making certain tests which consisted in filling strong glass globes with gases, sealing them tightly, and, by means of inserted wires, producing an electric spark therein, and igniting the gas. The considerable pressure thus generated proved too great for one of the globes, which exploded with great violence, many fragments of glass striking the experimenter in the face. The patient at once sought the services of a physician, who removed all those bits that he could find and gave him a solution of atropin for the eye. The latter, however, became painful, and these were the conditions I found:

A moderate, or light, plastic iritis with a tendency to adhesion inferiorly; an angular, valve-like wound through the cornea just exterior to the center, the two sides of the flap covering which would each measure about two millimeters.

Although the anterior chamber was not obliterated when the eye was first looked at, light pressure on the lids caused the aqueous to escape freely through the wound. A tiny abrasion of the anterior capsule of the lens directly behind the corneal wound, but absolutely no trace of any injury to the substance of the crystalline. It was evident that the same instrument which had punctured the cornea had pricked the anterior capsule. The most careful scrutiny failed to reveal the foreign body anywhere within the eye. The media were clear and the fundus normal with the exception of an area of œdema of the retina, occupying the center of the infero-temporal quadrant. The central vision was normal, but a blur was present in the supero-nasal field. Had a particle of glass or other material lodged within the eye and become lost to view, or had a needle-like piece punctured the cornea, jabbing the lens, and been withdrawn? All subsequent actions, or rather reactions of the eye, argued in favor of the first proposition.

The patient came to my office every day for one week. Meanwhile the eye had become quiet, the corneal wound had healed, and the iritis had disappeared. I had also made several exhaustive but vain searches for a foreign body. By the end of the week unmistakable detachment of the retina had occurred at the site of the primary œdema. The large vein traversing the area occupied a ridge from which its branches fell off like the veins from the stem of a wilted leaf—not flat and straight like those of a fresh, crisp leaf, as they should, and several grayish, opaque streaks appeared in the affected portion.

The patient was at once sent to the Passavant Hospital, put to bed and not permitted to rise from a recumbent posture on any account; both eyes were kept bandaged, and he was given daily hypodermics of the muriate of pilocarpine, increasing same until full physiological effects were obtained. After fourteen days of such treatment the retina was found to be re-attached. About this time another attack of iritis supervened, and again I made a careful search for a foreign body. I discovered a yellowish-white fungus-like growth on the iris near the bottom of the anterior chamber. On April 2d I called Dr. Hotz in consultation, and he inclined to the

belief that the foreign body was hidden in this growth. As a result, on the following day, April 3d, I made an incision with a keratotome straight downwards, an assistant holding a delicate cotton sponge just beneath the knife to catch the aqueous, together with whatsoever it might contain. With the first gush of the aqueous most of the fungus escaped; the rest was easily removed with forceps. No foreign body was found, although it was sought for with the aid of toilet-forceps and a delicate spatula. The eye recovered readily from the operation; but on the night before the patient was to leave the hospital, in a fit of nightmare, he threw himself from his bed and struck the bandaged eye on the corner of a glass table so violently as to open the keratotome incision and cause profuse bleeding. Singularly enough, when the eye cleared up—which it did very quickly—the retina was found undisturbed. The patient was sent home, but after a few days (April 19th, 1899) he came to my office with another attack of iritis *and with a bit of glass plainly visible in the anterior chamber.* Mr. G. stated that, on the evening before, he had gone down into the basement of his house to attend to the furnace, and that in attempting to look at the bars of the grate therein he had stooped forward, lowering his head almost to the floor; that in doing so he had felt a peculiar sensation in the eye as if something had given away.

Here then, after six weeks, was the foreign body which had been the cause of all our anxiety, and I resolved not to lose sight of it before making an attempt to extract it. I placed the patient in the operating chair at once, and, with the aid of Dr. E. L. Schwarz, set about it. I fancied that, by making an incision as before, if the glass did not come away with the aqueous I might be able to grasp it with smooth-jawed forceps. With the obliteration of the anterior chamber, however, the foreign body was caught tightly between the iris and cornea, even pushed down into the stroma of the iris so deeply that it was nearly lost in the excavation thus made, and resisted all attempts to remove it with forceps. I feared that a sharp angle would pierce the iris and anterior capsule, so taking the iridectomy forceps I quickly siezed the iris on either side of the depression containing the glass, withdrew it and made an iridectomy which included the glass.

The fragment was of rhomboid shape and measured 3 by 1 by $^{3}/_{5}$ millimeters. The eye recovered promptly and has remained healthy and quiet.

I last saw the patient in January of the present year (1901); the vision and refraction in the operated eye at that time were:

L.V. $=^{20}/_{30}$; $^{20}/_{15}$ w. + 0.5 c. ax 180°.

By ophthalmoscopic examination at this time the retina was found to be normal. The only indications of there having been anything wrong at the site of the detachment were a few tiny orange-colored spots, and a few crystals of cholesterine in the corresponding portion of the choroid.

Clearly, to my mind, the foreign body had been all this while in the posterior chamber. As to the manner in which it got into this chamber one can only surmise. It is barely possible that it passed through the iris, although there was no cicatrix visible, and, besides, the relative positions of the corneal and the capsular wounds argued against such a supposition. It may have stuck for a time in the anterior capsule, then, after the mydriasis from the atropin occurred, it might have let go and fallen behind the iris. Or it might have lodged first in the anterior chamber, and later, through some prone position of the head, have slid over the pupillary margin into the neighboring chamber. Certain it is, in my opinion, that there was where the foreign body lurked from the time I first saw the case until the evening before it was removed.

It is doubtful, owing to the small size of the particle, whether the Roentgen rays would have been of service in locating it, but these would soon have been resorted to had conditions not changed as they did. Yet, even had the position of the glass been thus determined, any attempt to extricate it from its original situation would have meant more traumatism and, possibly, disaster.

THE ESTIMATION OF THE DANGER OF SYMPATHETIC OPHTHALMITIS.*

By EDWARD JACKSON, M.D.,

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CASE I.—A woman, aged 60, came with a history that three years before she had suffered from violent inflammation of the left eye, which had lasted for nine months. The inflammation had followed a traumatism, but there was no evidence of a penetrating wound. Two years afterward inflammation had occurred in the right eye. In neither eye had there been any relapse or recurrence of inflammation. In neither eye was the tension diminished. The left eye, with the pupil dilated, counted fingers at two feet; the right eye at three feet. The right eye showed three broad, posterior synechiæ. The left pupil was bound down, except at the upper sixth of its circumference. This patient had consulted five ophthalmologists of large experience, three of whom advised enucleation of the left eye to prevent blindness of the right. The same measure had been strongly urged by two general practitioners who had charge of her case.

CASE II.—A man, aged 61, had suffered twelve years before from cataract of the right eye which followed typhoid fever. Early in January, during an attack of the grip, acute glaucoma occurred in the eye. The ophthalmologist called in did iridectomy under cocaine, and during the operation the hypermature lens "popped out upon his sleeve." Next day the eye showed some vision and continued to improve. But the corneal scar bulged, the eye continued sensitive and slightly painful, and the operator strongly urged that it be enucleated. Disagreement as to this brought the patient to me six weeks later.

I found the tension of the operated eye was not diminished, but it was at times somewhat increased. The bulging of the corneal scar seemed sufficient to account for the pain and hyperemia. There were no symptoms of sympathetic trouble in the other eye. At the end of three months the

*Read before the Denver Ophthalmological Society, April 13, 1901.

operated eye had, with a correcting lens, central vision of $^4/_{25}$ and a field of considerable practical value.

Case III.—A man, aged 42, came to me with the history that nine years before his left eye was lost by an accident while gunning. A shot was supposed to have penetrated the sclera at the nasal side and to have lodged in the eyeball. The eye had become free from inflammation in a short time, and had remained so, although its tension was $-T^2$. It possessed bare light perception. A week ago the right eye had become the seat of a violent iritis, which a colleague refused to treat unless he was permitted to enucleate the blind eye, evidently believing that he had to deal with a case of sympathetic inflammation. But there was no hyperemia, tenderness, or other disturbance of the blind eye. The acute iritis had not the special characters of a sympathetic inflammation, and had come on after excessive eye-work. I regarded it and treated it as a case of rheumatic iritis. The eye recovered with vision of $^4/_3$ partly, which was retained; and there had been no relapse seven years later.

These cases may serve as the text for what I have to say upon this subject. Tacitus long ago observed that the unknown is always magnified. In our ignorance of the essential nature of sympathetic ophthalmitis the possibility of its occurrence is calculated to arouse a terror of it, that is fatal to calm judgment. Among general practitioners this state of mind is quite common. We have all seen cases referred for decision as to the necessity of enucleation, where there was not the slightest reason to suspect a need for it. These of course must be considered along with the cases in which sympathetic inflammation is allowed to develop before the danger of it was seriously considered. Both classes are due to lack of acquaintance with the disease, or carelessness regarding its possible indications. But with the very best knowledge of it that we have and the most careful watching, there still remains an uncertainty regarding sympathetic ophthalmitis which is necessarily sometimes alarming. Cannot this uncertainty be somewhat lessened by a better understanding of a few cardinal symptoms that may serve as landmarks?

In the first place, sympathetic ophthalmitis follows injury.

But as a rule it follows only such injuries as cause penetration of the sclero-corneal coat with infection of its contents. I have never seen a case in which the history of such an injury was lacking. I know that a number of cases are reported as cases of sympathetic disease in which the injury was not attended with such perforation. But I do not know of any such case in which the course of the disease was at all the typical course of sympathetic inflammation. Anomalous cases should always be scanned most critically. And it is illogical to admit as a case of sympathetic inflammation, one that arises in an exceptional way, unless its symptoms and course are characteristic, and make it certain that no other explanation of its character is reasonable.

Secondly. Sympathetic inflammation does not follow mere injury of the exciting eye, whatever its character. If the injured eye wholly recovers it never excites sympathetic ophthalmia. Only when it remains permanently impaired and the seat of a distinct irido-cyclitis does it endanger the other eye. Among the symptoms of this special form of irido-cyclitis, diminished tension of the eyeball is so constantly present that its absence should almost rule out danger from sympathetic inflammation.

Third. The course of sympathetic inflammation is on the whole quite characteristic. It is marked by an insidious onset, the absence of which was an important fact in case 3 of the above series. It shows a strong tendency to temporary improvement and relapse. By the time the inflammation has become severe there is generally reduction of intra-ocular tension. In all cases of sympathetic inflammation there is organic disease of the uveal tract. This is speaking of the disease as we know it; a disease liable to cause blindness of the sympathizing eye.

Finally. The greatest confusion has sprung from lack of discrimination between sympathetic irritation and sympathetic inflammation. Under the head of sympathetic ophthalmia both are included, although the former is clearly not an inflammation of the eye, but a neurosis. That the essential pathologic condition of this neurosis may be a factor in causing sympathetic inflammation is quite possible. Still the common differences in the lesions of the exciting eye, and of

the time that has elapsed since the injury, seem to indicate that sympathetic inflammation and sympathetic irritation are quite distinct. At least this is clear, that sympathetic inflammation has its peculiar essential factor quite apart from the neurosis.

It would seem absurd to waste time in insisting on this distinction between sympathetic inflammation and sympathetic irritation were it not so persistently ignored, even in most recent literature. While preparing this paper there comes to hand *La Clinique Ophthalmologique* of March 25, 1901, in which Guibert reports four cases of "sympathetic ophthalmia"; one of which was a case of typical sympathetic inflammation, one of irritation without any inflammatory symptoms, and two anomalous cases in which sympathetic irritation was probably complicated by some sort of inflammation, but which were not cases of sympathetic ophthalmitis. Yet he proceeds to generalize upon them as though they were all instances of one and the same pathological condition.

A clear discrimination between sympathetic ophthalmitis and sympathetic irritation is of the greatest practical importance. The confusion of these two conditions is responsible for much of the uncertainty that still exists regarding sympathetic ophthalmia. A careful examination of the literature will show that most of the anomalous cases, the cases that confuse and blur the clinical picture of sympathetic ophthalmitis, really are cases of sympathetic irritation.

But sympathetic irritation is a condition about which we do not need to worry our patients or ourselves before it actually arises. No matter how severe or how long continued it may be, it can be cured by removal of the exciting eye at any time. Donders' case of restoration of normal vision in the sympathizing eye, after two years of blindness, within a few hours following the enucleation of the exciting eye, is an extreme yet typical instance. We are never compelled to do a prophylactic enucleation for sympathetic irritation. Indeed one is not justified in doing enucleation or its substitutes merely to prevent the possible occurrence of a condition that will be so readily and certainly curable if it should arise. The condition that we do have to guard against by prophylactic enucleation is one marked in every case by the organic

changes of inflammation, and, as I believe, always of inflammation involving the uveal tract.

Every report of a case of "sympathetic ophthalmia" which does not make clear that the case does or does not belong to this class merely adds to our confusion and thus is harmful. It is certain that most cases of sympathetic disease, arising long after the injury of the exciting eye, or from non-perforating injuries, or which are excited by eyes that have undergone degenerative changes without traumatism, are cases of sympathetic irritation. Of the same nature are most of the cases that arise after the exciting eye has become quiet and free from tenderness. But we cannot, from the literature, feel sure that all cases that arise under such circumstances are of this less dangerous character.

When this confusion of sympathetic irritation and inflammation has been cleared away, and our ideas concerning the latter are rendered more definite, we shall probably be able to distinguish very clearly the clinical picture of sympathetic inflammation, and the exact conditions that are likely to be followed by it. Even now a sufficiently accurate estimate of that danger may be formed to avoid hastily condemning to enucleation cases like those narrated above. Such an estimate should also prevent the sending out into the community of patients who, after being told that they would "go blind" unless they submitted to enucleation, have lived for many years without going blind, and without suffering the annoyances of an artificial eye. Patients with such a history teach their friends how little surgeons know about the matter; and ultimately cause blindness by leading some one to refuse enucleation when it is really needed.

PAMPHLETS RECEIVED.

"The Electric Skiascope," by H. Wolff, M.D.

"Self-Examination for Medical Students." P. Blakiston's Son & Co.

"Choroiditis and Retinitis Centralis Serosa," by H. Wolff, M.D.

"A Rule for Combining Crossed Cylinders," by H. S. Pearse, M.D.

OPTIC NEURITIS FROM INTRA-NASAL DISEASE.*

By DERRICK T. VAIL, M.D.,

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THE thin shell of bone separating the orbital tissues on the one side from the nasal structures on the other has subserved the purpose of a true and impervious boundary wall between the eye and the nose.

The structures, functions and purposes of the eye and its auxiliary tissues are so very different from those of the nose that some oculists disdain such an apparently alien neighbor as the nose; and, on the other hand, a few rhinologists boast of knowing as little of the eye as of the bones of the foot.

A true boundary between the eye and the nose does not in reality exist; on the contrary there is a very intimate connection. Beside mere contiguity, there are abundant and open nervo-lympho-vascular relationships, so that we should regard the innervation, the lymph and blood circulation of the eye and those of the nose together and not apart. The nasal branches of the trigeminus nerve supplying the ethmoidal, sphenoidal and frontal sinuses are offshoots of the ophthalmic branches of the same nerve lying within the orbit. The arterial supply and the venous drainage of the upper accessory sinuses are really part and parcel of the ophthalmic system of arteries and veins. The lymph channels of the nasal attic drain into the orbit where they anastomose with the perivascular, perineural and peribulbar spaces; anatomical points which should open our eyes to the fact that the upper pneumatic cells of the nose and the important soft tissues of the orbit are intimately and inseparably associated.

Kuhnt says: "The orbit is surrounded on three sides by accessory nasal cavities lined with mucous membrane, the veins from which empty directly into the ophthalmic veins."

Since this paper deals more particularly with the sphenoidal cell in relation to optic neuritis, it will do no harm to make a tour of inspection of this cavity. After considerable difficulty we find the narrow opening leading into the cavity of the

*Read at the meeting of the Western Ophthalmologic and Oto-Laryngologic Association, held in Cincinnati, Ohio, April 11-12, 1901.

sphenoidal body. We crawl through the small entrance and drop inside. It is dark, stuffy and quite unattractive. The single window admits of no air current, there being no counter-opening. Its irregular walls are lined with glistening, thin and pale mucous membrane, which is almost entirely devoid of glands. We hear the ominous and rhythmical rush of arterial blood as it courses through the large vessels lying just beyond the confines of the cavity. We note the low musical whirr of venous blood circulating through the ophthalmic vein and the cavernous sinus. The quiet nasal respiration of the Gulliver whose sphenoidal caves we Lilliputs are exploring, sounds like the mighty roar of a tornado. The cavity echoes and augments the sound. The guide points to the irregular ceiling and tells us that just on the other side of the thin covering of the sphenoidal cell lies the optic chiasm. He touches the outer wall near the ceiling with his Alpine stick and announces that the optic foramen is there, and that the optic nerve at that point lies against the cell-wall itself. He tells us that the wall here is exceedingly thin and in some cases membranous. He warns us not to lean on the outer wall, for we might compress the ophthalmic vein or tickle the fifth nerve, or irritate the third, fourth or sixth nerves which pass just on the outside.

We perceive at once that if this gloomy, stuffy, illy-ventilated cell should become infected with pus-formers, resulting in abscess, or even if it became the seat of a less violent but infective inflammation, the important outlying ocular structures would be in real jeopardy. In the former case the lateral wall of the cell might become swollen or even bulging. Perhaps there would be more or less pinching of the nerves and vessels passing through the optic foramen and the sphenoidal fissure. A neuritis from compression would result. In the latter case some of the infection existing in the stroma of the mucous membrane lining the ethmoidal and sphenoidal cells might be taken up by the venous and lymph circulation to be carried into the larger channels of the orbit. This would mean infection and swelling of the orbital lymph spaces, sluggish circulation in those spaces and even stasis, and the subdural lymph space of the optic nerve would, in some cases at least, become affected. Thus a true interstitial optic neuritis

might result. Pain in and around the eye would in many cases be a prominent symptom, due either to direct compression of the ophthalmic division of the fifth nerve where it passes through the sphenoidal fissure, or reflexly from the sinusitis via the anterior and posterior ethmoidal nerves—branches of the ophthalmic. Septic matter taken up by an open vein may be carried from the nose into the veins of the orbit, where in certain cases septic thrombosis would result. Hajek points out that even the cavernous sinus has become infected from the upper nasal veins. Thrombosis of the orbital veins has resulted after nasal operations where sepsis of the wound occurred and even where the galvano-cautery was used on the turbinates.

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We all know how common acute coryza is. We recognize and expect it to be an accompaniment of grip, measles, whooping-cough, etc. We are familiar from personal experience with the distressing nasal and head symptoms, and often remark on the profuse rhinorrhœa that indicates the "breaking up" of the cold.

Modern authorities claim that sinusitis very frequently exists in those cases. The causes for acute sinusitis mentioned in the new text-books (Shurly, L. Brown, P. Brown, Kyle, *et al.*) are influenza, the acute exanthemata, infection from nasal operations, the retention of secretions from nasal packings, etc. Grip is conceded by all to be the most potent cause. Frankel, of Hamburg, found acute sinusitis to be present in *all* of one hundred and forty-six patients dead from grip. In not a single case was sinusitis diagnosed. Weichselbaum, of Vienna, performed many post-mortem examinations on patients dead from grip and makes the statement that 90 per cent. had sinusitis.

It is fortunate that the front wall of the sphenoidal cavity is usually the thinnest and gives way first, otherwise acute retrobulbar neuritis and blindness would be much more common.

I leave it to an abler pen than mine to tell us how to make a positive diagnosis of *acute* sinusitis. I frankly admit that I am anxious and willing to be taught both how to recognize it and how to cure it.

At any rate we must concede that acute empyema of the ethmoidal and sphenoidal cells does occur as well as necrosing ethmo-sphenoiditis, and from that concession we can safely argue that we have a well-founded cause for acute retrobulbar neuritis and all the alarming accompaniments.

If rhinologists will grant that pus confined within the cells of the ethmoid and sphenoid would cause inflammation, thickening and bulging of their orbital walls or infection of their lymph spaces, oculists will, I think, be able to produce a *causus morbi* for certain curious and acute retrobulbar inflammations affecting the optic, the fifth and sometimes the third and sixth nerves.

In case of erosion, thickening or bulging of the lateral wall of the sphenoidal cavity, in certain cases we would have at first compression of the optic nerve and some choking of its foramental part. This would be resented by the soft and easily insulted nerve fibers and their fine capillaries and lymph spaces, and the result would soon be a true interstitial neuritis with some distention of the vaginal sheath in front of the optic foramen (and not behind it in the chiasm). The neuritis would extend toward the ball, since it would be the distal end which would suffer from such strangulation. In the phenomenon which follows putting a tight rubber band around the finger, all the swelling is on the distal side of the rubber, and so in case of strangulation of the optic nerve at the apex the inflammation would extend forwards, thus producing a monocular optic neuritis. Other stages would follow in rapid succession—transudation, plastic exudation, complete arrest of function, and finally atrophy and fibrosis, which would obliterate the finer markings of the nerve, destroying endoneurium, perineurium, and neuraxons alike.

The question might here be asked: "In acute retrobulbar neuritis, is there a true choked disc?" The answer is, "No, the artery and vein of the disc enter the optic nerve only about 10 or 15 mm. behind the globe; at that point there is a free enough inlet for arterial blood and an outlet for venous blood to prevent that." The throttling of the optic nerve is at the foramen, an inch farther back.

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A certain quiet type of monocular optic neuritis we all have seen over and over again. Hitherto no explanation of the disease has been offered. We note the presence of a distinct exudative neuro-retinitis in one eye only and the entire absence of a systemic cause. The subjects are usually under twenty-five years of age and in apparently robust health. The disease is "quiet" in that there is usually no involvement of the fifth nerve and the amblyopia is gradual and never complete. The vision falls to $^{20}/_{200}$ or less, and then there is a clearing up of the optic nerve exudate and with it a gradual return of sight. Usually there is no atrophy of the disc resulting, and only a slight lack of definition at its margin or a small bunch of organized lymph along one or two of the vessels coming out of the disc for a short distance beyond its border. In the four cases of this class which I had during the past year the patients had had a short time before a mild attack of "cold in the head on that side," or of grip. Their only symptom was foggy vision. They all recovered perfect sight by the usual treatment. In these cases a balloon swelling of the turbinates, which disappeared of its own accord after the eye recovered, was all that was noted in the nose. The writer does not wish to claim that every one-eyed optic neuritis is of nasal origin; he merely wishes to give the nose the amount of consideration which its intimate relationship with the nerve and perineural structures seems to demand.

We may have three distinct types of optic neuritis from nasal origin:

(*a*) *Acute fulminating retrobulbar neuritis*, due to mechanical compression of the optic and ophthalmic divisions of the fifth nerve from swelling of the lateral walls of the sphenoidal cavity. This form results in profound rapid amaurosis associated with violent cyclonic neuralgic pain.

(*b*) *Acute retrobulbar perineuritis and optic leptomeningitis*, due to infection (?) carried from the nose via the lymph channels. Here there is no compression neuritis like in the foregoing class, but there is infection and swelling of the subvaginal and perineural lymph spaces. It is manifest that this class is free from pain and that the amaurosis is much less marked, although the ophthalmoscopic picture is much the same as in class "*a*."

(*c*) *Retrobulbar optic neuritis secondary to septic venous thrombosis*, the origin of which is intra-nasal in a vast majority of cases.

In the acute fulminating retrobulbar neuritis the symptoms are profound and unmistakable. The patient first notices twinges of pain in eyeball, brow and temple. Pain soon becomes unbearable in some cases. The eye becomes slightly congested, suffused with tears, rotation painful, pupil slightly dilated and very sluggish. Tension normal, vision very bad, soon becoming entirely or nearly abolished. At first only slight hyperæmia of the optic disc is noticed, soon followed by marginal indistinctness, due to filmy exudate. Veins now become overfilled but not tortuous; optic nerve slightly swollen, generally less than 1 D. The exudate extends a short distance into the retina surrounding the nerve-head. The disease runs a very acute course. The pain soon subsides; the vision begins to return in from three to ten days after the onset; the exudate disappears. Daily tests of the visual field show rapid return of whole segments. The center of the field is slow in returning; in some cases central scotoma remains permanently. The optic nerve now becomes distinctly atrophic, especially on the temporal side of the disc, representing the papillo-macular bundle of fibers; the blood-vessels are not much, if any, reduced, and finally in favorable cases there is complete restoration of sight. The above symptoms are culled from my notes of a case I had nine months ago in which necrosing ethmoiditis and sphenoiditis were positively present. The optic nerve atrophy, which is usually but partial, remains unchanged for the balance of the patient's life.

Deyl (in Norris and Oliver) states: "Acute retrobulbar neuritis is characterized by its sudden and fulminating inception, generally accompanied by severe pain in eye, back of eye, forehead and head; rapid and marked disturbance of vision amounting to blindness in from one to several days and sometimes in a few hours," but he makes no mention of a possible nasal origin, excepting that it is due to "influenza" in many cases.

Elschnig says: "The lesion is primary interstitial neuritis —catching cold the only assignable cause."

De Lapersonne (*Knapp's Archives*, 1900) declares optic

neuritis of this kind to be due to ethmoid and sphenoid disease. He states that the eye symptoms draw our attention to the sinus disease that had been overlooked. He makes use of the following words: "The most frequent ocular symptoms of sphenoiditis is a retrobulbar neuritis *localized in the optic canal.*" He reported three cases of unilateral acute retrobulbar neuritis which led to the discovery of extensive pathological changes in sphenoidal and posterior ethmoidal cells.

Berger says: "Optic neuritis may develop from acute infective disease in the nasal accessory sinuses."

Hansell, of Philadelphia (*Med. and Surg. Rep.*, 1896), reports a case of blindness from ethmoidal and sphenoidal disease. The nasal disease was distinctly present and the diagnosis seemed perfectly clear.

Hajek, in his classical work on the nose, states: "If the bony framework of the sphenoidal cell is involved from sinusitis, serious complications result, rapid blindness—monocular, has been observed many times. The cause is either pressure against the optic nerve at its orbital entrance, or perineuritis, each meaning that the bony wall has been eroded."

Dr. C. R. Holmes, of this city, reported in *Knapp's Archives of Ophthalmology*, 1896, a clear case of acute retrobulbar neuritis, due to sphenoiditis, presenting all the classical symptoms and recovering after a considerable quantity of pus had been liberated from the sinus. He illustrated his complete report by anatomical drawings from sections he had made. I had the pleasure and opportunity of working with him throughout that case, which I believe was the first of its kind published, demonstrated and explained. But if you will scan the literature from the time Helmholtz gave us the ophthalmoscope down to the present, you will find reported dozens and dozens of cases of acute blindness in one eye associated with the ocular symptoms mentioned, and in a very few you will find mention made of the nose. This statement stands true up to the present day. The object of my essay is to call attention to the fact that the nose is usually not thought of in this disease. The unfortunate circumstance in the make-up of this naso-ocular disease is the fact that rhinoscopy usually fails to reveal more than a slight and apparently simple acute boggy swelling of the turbinates. The ocular symptoms entirely overshadow the nasal.

In the diagnosis of acute retrobulbar neuritis we would have to exclude all other diseases and conditions, such as glaucoma, orbital neoplasms, cerebral syphilis, brain tumors and abscesses, poisoning from lead, quinine, tobacco, stramonium, iodoform and the other toxic substances, diabetes, albuminuria, malaria, anæmia, chlorosis, specific neuroretinitis, embolism of the central artery, and hysterical amblyopia. In the presence of any of these in a given case the diagnosis of optic neuritis from endorhinitis would hardly stand.

The striking features of acute fulminating retrobulbar neuritis from nasal origin are:

(*a*) The rapid onset, course, duration and termination as compared with other forms of optic neuritis.

(*b*) The presence of excruciating pain, involving most all of the branches of the first division of the fifth nerve.

(*c*) The complete loss of vision in a short space of time after the onset (from a few hours to a few days), and the rapid return of fair vision in most of the cases with, however, more or less optic nerve atrophy and frequently with absolute or relative central scotoma.

(*d*) The absence in many cases of positive evidence within the nose that the trouble is of nasal origin.

The treatment will suggest itself. Inasmuch as blood-letting is indicated anyway, I would not hesitate to remove all or a part of the middle turbinated bone and at least try to get into the sphenoidal cavity, but would be heedful of the dangers. I think the middle and superior straits of the nose are the ideal places from which to draw blood in this class of cases, because the opening of blood-vessels supplying the nasal attic would have a direct influence on lessening the orbital congestion.

The nasal operation affords a two-fold purpose—blood-letting and drainage.

MEDICAL SOCIETIES.

PROCEEDINGS OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM.*

Thursday, March 14th, 1901.

G. Anderson Critchett, M.A., F.R.C.S.E., President, in the Chair.

The Evolution of the Color Sense.

Dr. F. W. Edridge-Green read a paper on this subject. He said the facts of color blindness threw a good deal of light on the evolution of the color sense. Cases of color blindness might be divided into two classes quite separate and distinct from each other, though both might be present in the same person. In the first class there was light as well as color loss. In the second class the perception of light was the same as in the normal sighted. Many color-blind persons belonging to this class had a light perception much more acute than was found with most persons having a normal color perception. Color-blind individuals belonging to this class could be arranged in a series. At one end of this series were the normal sighted, and at the other end the totally color blind. The author had classified them in accordance with the number of colors which they saw in the spectrum, as pentachromic, tetrachromic, trichromic, dichromic, and monochromic. All the facts which could be gained from the study of museums and literature pointed to the view that the sense of light was developed first, and then the sense of color, those waves which differed from each other most physically, being first perceived as different—namely, red and violet. It was obvious that if a certain portion of the visual center were set apart for the perception of color, when it was very small and ill-developed, it would only be able to perceive differences of considerable magnitude; but as more cells were added to the center greater and greater power of perceiving differences arose. Each cerebral cell might at first have been connected with many optic nerve

*British Medical Journal.

fibers, and the number gradually diminished as evolution proceeded. It need only be assumed that the nerve impulses differed in quality, for instance, in wave length, just as the waves of light differed physically in wave length, and that when a nerve impulse of a certain character acted on a cerebral cell it gave rise to a corresponding sensation of color, within the range of perceiving differences possessed by that cell. It was obvious that if the nerve impulses did not differ in quality one could never have a sensation of color. The spectrum was, therefore, first seen as grey, then with a tinge of red at one end, and violet at the other. As the color sense developed it was not necessary that the rays should be so far apart before a difference was seen, and so the grey band gradually diminished until it entirely disappeared and the two colors met in the center of the spectrum. A third color, green, then appeared at the central point. Then yellow appeared, making four colors; then blue, making five; and finally orange, making the normal six. In some persons evolution had proceeded further, and seven colors were seen, there being two colors between the green and the violet, instead of one. It was found that paintings were first monochromatic, then red and violet were used as colors, then green, then yellow, and then blue. Homer's color vision was of the degree just preceding total color blindness. The description by different authors of the rainbow as having one, two, three, four, five, six, or seven colors was very characteristic. These facts showed that color blindness was only an example of an earlier state in the development of the color-perceiving center.

Peritomy in Episcleritis, Iritis, Herpes Ophthalmicus, and Purulent Ophthalmia.

Mr. T. Pridgin Teale read this paper. He traced shortly the history of the operation, which was modified from syndectomy, in which a portion of the conjunctiva was excised. This, however, was found unnecessary, and if a greater effect was desired than that produced by cutting the conjunctiva round close to the cornea, it could be obtained by making several radial incisions in it. Mr. Hewetson, of Leeds, drew attention to the value of the operation in interstitial keratitis which the author fully endorsed. He wished to point out its great value

in the other diseases mentioned, and especially in episcleritis, in which it was most marked, the pain being greatly modified. The same was noticed in obstinate cases of iritis from any cause. In a case of subacute purulent ophthalmia with ulceration of the cornea in a child a complete cure resulted. He had also performed the operation in a case of irido-cyclitis with increased tension where there were reasons for not performing iridectomy. In this case the operation reduced the tension to normal, and gave great relief, though it did not cure the disease.

The President said he usually removed a small ring of conjunctiva, and often with excellent results.

Mr. Cartwright thought the good effect produced was by its acting as a counter-irritant, and not in a mechanical manner.

Mr. Secker Walker had seen good results in interstitial keratitis, and also in iritis, and he looked upon its action as being similar to that of a leech.

Mr. Hern said he removed a piece of conjunctiva, and curetted round the wound. He felt it was a much-neglected operation.

Remarks were also made by Messrs. Marcus Gunn, Johnson Taylor, and Cropper.

Membranous Conjunctivitis.

Mr. J. F. Bullar described a case. The patient was a child, who was born on October 28, 1900, and who was quite well when seen on November 5th. On November 7th the right eye was noticed to be affected, and was treated by the nurse until November 11th, when it became much worse, and the doctor was called in. When Mr. Bullar first saw it the lids were very much swollen, a yellowish membrane was present, which stripped off easily and left a slightly bleeding surface. Nitrate of silver gr. ij ad ℥j was used. The eye got better, the chemosis subsided, leaving a small central ulcer of the cornea. Membrane was also noticed on the right side of the nose and fauces, which extended. The child died in convulsions on November 20th. There was no diphtheria in the neighborhood, and the membrane was not examined.

Mr. Sydney Stephenson made it a rule to use antitoxin

on first seeing a case, without waiting to verify the diagnosis bacteriologically.

THE PRESIDENT said Mr. Tweedy, who had had great experience in these cases, looked upon quinine as being a specific in diphtherial conjunctivitis, and he had himself seen great benefit arise therefrom.

MR. LANG said that Mr. Tweedy's observations had been made long before antitoxin was known, and he thought it should not go forth as the opinion of the Society that his view was still held.

MR. DEVEREUX MARSHALL said he should not think of waiting to conduct a bacteriological examination (though this would not take long), but in a case of supposed diphtherial conjunctivitis he should at once inject antitoxin, which in no case could do harm, and in a true case the sooner it was done the better. With regard to the local application of quinine, he strongly approved of it, though he had not seen its use followed by such excellent results as those obtained by Mr. Tweedy.

OTHER PAPERS.

Two papers by MAJOR H. HERBERT, I.M.S., were taken as read. The first was on hypertrophy and ossification of the tarsus. A male Hindoo, aged 33, had considerable enlargement of both upper tarsi, especially towards the outer ends, without much thickening. On the left side it was fibrous, but on the right there was considerable bone formation, which was fairly dense. A piece of necrosed bone exposed on the conjunctival surface of the lid kept up irritation, for which the patient sought relief.

The other paper was a note on the surface of the normal cornea. Over thirty normal corneæ were examined in Bombay, all of which showed more or less epithelial defect when the surface was partially dried by exposure, the upper lid being elevated from the eyeball, and local illumination and the corneal loupe being used. In some cases there were only one or two pits seen, in others many closely-set pits, in others grooves added, in others one or more large depressed patches. The depressions were all sharply defined, and their floors stippled owing to the irregular surface presented by the deeper

epithelial cells. They usually began to appear within half or one minute, sometimes within a few seconds, but were generally not fully shown for two or three minutes. Repeated exposures and the instillation of cocaine sometimes caused an extensive irregular superficial exfoliation of the epithelium, which partly obscured and altered the original picture. The patients examined were all native, mostly adults. Slight conjunctivitis and localized keratitis tended to exaggerate these physiological defects. In superficial punctate keratitis some of the points stained with fluorescine appeared depressed on drying and had to be distinguished from the physiological pits above described, possibly to be seen on the same cornea.

Card Specimens.

The following card specimens were shown: Mr. Hartridge: (1) A Capsulotomy knife; (2) Case of Central Choroiditis Simulating a Neoplasm, which had been watched for fourteen years. Mr. H. Secker Walker: A Diagrammatic Model to Assist in the Teaching of Refraction. Mr. J. H. Tomlinson: A New Color Perimeter. Mr. J. H. Parsons: Coloboma of the Optic Nerve. Mr. G. W. Roll: Immobility of the Pupils with Fundus Changes. Mr. P. Flemming: Section Showing Dilated Nerve Fibers in the Retina. Mr. Doyne: A Simple Form of Ophthalmoscope. Mr. Work Dodd: A Case of High Hypermetropia.

The following officers were elected at the sixth annual meeting of the Western Ophthalmologic and Oto-Laryngologic Association, held in Cincinnati, April 11th and 12th: Dr. C. R. Holmes, Cincinnati, Ohio, President; Dr. W. L. Dayton, Lincoln, Neb., First Vice-President; Dr. J. O. Stillson, Indianapolis, Ind., Second Vice-President; Dr. H. W. Loeb, St. Louis, Mo., Third Vice-President; Dr. O. J. Stein, 100 State St., Chicago, Treasurer; Dr. Wm. L. Ballenger, 100 State St., Chicago, Secretary. At the meeting in Cincinnati the scientific program was of very high grade. Forty new members were elected. The next meeting will be held in Chicago, April 10, 11 and 12, 1902.

ABSTRACTS FROM MEDICAL LITERATURE.

By W. A. SHOEMAKER, M.D.
ST. LOUIS, MO.

EXPERIENCES IN THE USE OF HOMATROPINE AS A CYCLOPLEGIC.

Edward Jackson (*Annals of Ophthalmology*, January, 1901) considers homatropine, if properly used, a reliable and satisfactory cycloplegic, as reliable in children as in adults. His favorite method of using it is to instill one drop of a 2.5 or 3 per cent. solution every five minutes, five or six times. It rarely produces any toxic symptoms.

THE USE AND ABUSE OF SPECTACLES.

Hasket Derby (*The Boston Medical and Surgical Journal*, February 28, 1901) thinks spectacles are frequently prescribed when they are not absolutely necessary. In moderate degrees of hypermetropia it is a mistake to use glasses for anything but close work, for the patient, through constant use, becomes dependent upon them. In myopia glasses are probably too little worn, but great care must be exercised in treatment and no glass should be ordered until a thorough investigation of the history of the patient, the family tendencies, the acuteness of vision, and the condition of the interior of the eye has been made. As to astigmatism, so much relief has been given by glasses that the tendency is to prescribe them when they are not really needed. The author believes they can be dispensed with in cases in which vision, either with or without a spherical glass, is found equal. As to heterophoria, it is often cured by an improved general condition without the use of prisms.

L'ACCOMMODATION DANS L'OEIL HUMAIN.

Wilhelm Schoen (*Archives d'Ophtalmologie*, February, 1901) attacks the Helmholtz theory of accommodation. He says it is not sufficient from an anatomical, physiological or pathological point of view, and he presents a new one in place

of it. As everybody knows, in accommodation for the near, according to Helmholtz, there is a relaxation of the zonule, and for the far it is tense. Schoen repeats the experiments:

First, as to the movements of the ciliary processes.

Second, to the movement of the Purkinje images.

Third, the difference and the change of tension.

Schoen says that the test as to the mechanism of the accommodation is extremely difficult and always susceptible of an equivocal interpretation, although the anatomy does not exhibit difficulties. There is great diversity of opinion as to the course of the zonular fibers, yet the principal facts are now well established, and we understand the zonule perfectly. The course of the fibrils is to-day well known.

1. Each zonular fiber makes a part of a cell of the ciliary epithelium or of a cell equivalent to that of the system of nutrition of the retina.

2. In fact, all the cells of the ciliary epithelium and of the zonular fibers represent a kind of prolongation of the retina, which is a prolongation up to the capsule of the crystalline lens.

3. In front of the ciliary process there is an interlacing of the zonular fibers.

4. All the zonular fibers which are attached to the anterior capsule of the crystalline lens take their origin from the margin of the retina and the posterior part of the ciliary epithelium *passing behind the ciliary processes, without being connected with them.*

5. The fibers springing from the anterior part of the epithelium are attached to the posterior capsule much more rarely than the others.

6. The tendons of the meridional fibers of the ciliary muscle are situated in the external layers of the choroid, without having any organic communication with the internal layers of the choroid and still less with the retinal margin of the ciliary epithelium, where the zonular fibers have their origin.

7. Never, or scarcely ever, is the lens placed symmetrically at the angles of the ciliary body.

8. From the original retinal margin up to the angle of the ciliary body and farther beyond, the limiting membrane loses itself between the two layers of the ciliary epithelium of the

pigment. The adherence between the two layers is so intimate that they cannot be separated without injury, but when that limit is reached the retina is easily detached from the choroid, because between them is found the space of the primitive ocular vesicle.

The theory of Helmholtz, continues Professor Schoen, is a simple hypothesis, but it is shown to be defective by the anatomy in three ways:

First, the circular fibers of the ciliary muscle have not yet been discovered. The theory does not apply to the meridional fibers.

Second, his theory supposes that the meridional fibers pass in front of the posterior extremities of the zonule, which is impossible, because these fibers do not attach themselves to the point of origin of the zonule.

Third, the conception of Helmholtz as to the zonule is completely erroneous. He describes one membranous fold, which attaches itself to the capsule in a marvellously sinuous way. Schoen claims that the theory of Helmholtz is also opposed to physical laws. Helmholtz claims that the tension of the zonule keeps the crystalline lens flattened.

The posterior extremities of the zonule are not fixed to the tissues of the sclerotic like cramp-irons in the walls of a house. Again, the fibrous membrane does not possess the rigidity of the capsule, and the extremities in question do not touch the sclerotic at all. In reality it is the tension of the vitreous body that holds the zonule and prevents it from plating and from relaxing. According to the theory of Helmholtz, the tension of the vitreous body during the absence of accommodation should be greater than that of the anterior chamber. Again, the theory of Helmholtz does not allow us to explain in the act of accommodation its effect—that is to say, the increase of the tension in the vitreous body over that of the anterior chamber, which should cease. The contents do not lose in mass, and the circumference does not increase the traction carrying forward the posterior surface of the zonule. It cannot diminish the tension of the vitreous body or relax the zonule. It would rather augment the pressure of the vitreous body on the posterior surface of the crystalline and of

the zonule, as much as the tension of the posterior of the crystalline is not diminished.

According to the theory of Professor Schoen the mechanism is entirely simple. He has already explained that the zonule represents a prolongation of the retina. The external capsule of the crystalline lens, the zonule and the retina constitute the envelope of a ball in which is contained the vitreous body and the crystalline. The meshes of the zonule are closed by the hyaloid membrane. This ball—which may be compared to an India rubber ball filled with a liquid—is surrounded by the fibers of the ciliary muscle and its processes, except at the anterior part. If one presses between the two hands a rubber ball and leaves free only a small part in exercising a concentric pressure the free part will be pushed forward. This apparatus can be still more exactly imitated by holding the ball between the two palms of the hands in such a manner that the portion not grasped will represent the ciliary processes. The contraction of the circular fibers presses the ciliary process against the circumference of the ball, the zonule, which makes the free part project—that is to say, the anterior capsule against which the crystalline is situated.

The meridional fibers prevent the ciliary processes from gliding on in advance. This suffices for the physiological accommodation. In the forced accommodation the meridional fibers press the ball, but in general their only function is to support the tension of the interior of the eye. Besides this, they furnish a tonic innervation. When the eye is healthy the intra-ocular pressure is not exercised on the sclerotic. Schoen claims to have simplified the model of the mechanism of accommodation. The great difficulty to be overcome was to reproduce the hydrostatic action of the vitreous body.

The details of the apparatus are easily understood. The posterior fibers of the zonule are relaxed during accommodation. After having recovered their tension until the crystalline has returned to the state of repose, they unite in smoothing out the crystalline.

The Theory of Tscherning and the Anatomy.—Tscherning admits with Schoen the increase of the tension of the zonule in accommodation for the near, but he gives a different explanation. He supposes a traction from behind exercised

by the ciliary processes. That proposition is contradicted by the anatomy, because the ciliary processes have not the least connection with the zonular anterior fibres, and they are incapable of exercising such attraction. We cannot apply this theory to the natural condition where the posterior extremity of the zonule fixed by the cramping irons is carried outwards.

As has been said, all experimentation on the mechanism of the accommodation is extremely difficult. It is almost impossible to obtain an immobility of the eye sufficient to destroy the influence of the pulse and the respiration. The more so, because the small variations in convergence do not allow the production of the accommodation between them. On the other hand, the things that one ought to observe—that is to say, the movements of the images of Purkinje or of the points of the ciliary processes—are extremely small. It is readily understood that the magnitude of the instruments used and the manipulations make considerable errors possible. This being so, one is not astonished that two scientists of equal merit, making the same experiments, come to diametrically opposite conclusions. As soon as the accommodation commences to act, the ciliary processes approach the crystalline, according to Becker, but recede, if one is to believe Coccius. That is then in reality an effect of perspective. Then Cramer admits that during the accommodation the image of the anterior surface of the crystalline diminishes and is further forward, an opinion adopted by nearly all the others. Monoyer proves that the opinion is not exact. If the image seen on the side appears to advance and that of the other goes behind, that is then also an affair of perception. The position of the crystalline axis has changed, but the image has not advanced. Very lately Tscherning has modified the opinion which he first announced. Then he thought he had found a different tension in the anterior chamber and in the vitreous body. My experience agrees with that of Hess, who says that it is impossible to prove a difference of tension between these two spaces during the accommodation. Undoubtedly these experiments may be modified more or less, but the results given by them do not disagree with physical or anatomical conditions. Certainly one may doubt the result of the expe-

rimenter who asserts that the refraction and tension of the eye are not changed after death, seeing that one is able without difficulty to perform an operation for cataract on the eye of a cadaver, with such an appearance of reality, notwithstanding it has lost its resisting power. Against the assertion advanced in the second place, that the refraction is augmented after the section of the zonule fibers, one may observe that the operation is so great that it is not possible to know anything of the physiological condition, and that it is certainly impossible to arrive at a sufficiently exact conclusion.

The results of all experiments mentioned up to this point are claimed to be compatible with Schoen's theory, and perhaps also with that of Helmholtz. The action of the anatomical apparatus is such that it should be accompanied by a symmetrical oscillation of the crystalline, and experience has proved its existence. The play of the accommodative apparatus, according to our theory, is always in intimate dependence upon the ciliary process, which are comparable to the ends of the fingers. That which allows this change, not only in the direction of the axis and the place and the form of the crystalline, as for example, the smoothing out of the lateral parts, has been demonstrated by Tscherning. The conception of a visual line is useful for the dioptric calculation, but it cannot be considered as an anatomical reality. It is not probable that the position of that line remains the same during accommodation. That oscillation compatible with an increase in the tension of the zonule is produced by the tremulousness which usually accompanies the forced contraction of a muscle. This should not be confounded with the oscillation of the crystalline, which is observed in the subluxation, and does not at all signify a relaxation of the suspensory ligament. Helmholtz did not seek to prove the truth of his hypothesis by these experiments. One is forced, therefore, to improve and to refute Schoen's theory at the same blow, without embarrassing the others in their anatomical objections. The experiments undertaken to sustain a preconceived opinion are always in danger of being badly conducted. In this case is the attempt to prove that the zonule is held in a state of repose and relaxation during accommodation. Hess admits, like the present writer, that there is no difference during ac-

commodation' between the tension of the vitreous body and that of the anterior chamber, undoubtedly because he believes that state would be in accord with the theory of Helmholtz. This is an error, for that theory demands in advance of accommodation, a tension stronger than that of the vitreous body and during accommodation augmentation of the tension in the anterior chamber or diminution of the tension of the vitreous body, until equilibrium exists between them. Since experience goes to prove that the crystalline descends in following the law of gravitation during accommodation, there will be many objections to the equivocal character of such an experience, for example, the rotation of the eye around the visual line joined to the movement of the head; but I do not insist, because this experience proves exactly the contrary to that which is deduced by M. Hess. The accommodation, in the sense of the theory of Helmholtz, as we have said, cannot press the vitreous body more strongly on the posterior surface of the crystalline and of the zonule. The tension will be consequently augmented and not decreased. The crystalline will then be made consequently more strictly immovable than ever. On the other hand, the idea of downward movement is not in want of accord with Schoen's theory, because as in our ball mentioned above, the crystalline of the vitreous body reforms and constitutes the kernel or nucleus of the eye. It becomes more contracted in the state of accommodation than in that of repose, and encounters for that reason a less difficulty in sinking down than in the first or second case, and in short, Schoen insists on the difference between the theory of Tscherning and his, that his antagonists are constantly contradicted. They are not able to relieve themselves of an error many times repeated, that the crystalline will be attached to the sclerotic in a certain manner like the lining to a stiff box. That admitted, Schoen claims, "the attachment should be more close during accommodation than during repose." The theory of Helmholtz, accepting one or the other alternative, remains Schoen's theory or that of Tscherning. But there exists a third alternative represented by Schoen's theory. According to the theory of Tscherning, the contact should be more intimate during the act of accommodation. That theory rests on the depression of the crystalline under

the influence of its weight. That observation is exact, and Schoen says that his theory is not affected by it.—*The Post-Graduate*, April, 1901.

NOTE ON THE PECULIAR NYSTAGMUS OF SPASMUS NUTANS IN INFANTS.

John Thomson (*British Medical Journal*, March 30, 1901) says: In most cases of head-shaking, or spasmus nutans, in young children there is nystagmus. The close relation between this symptom and the head movements has often been pointed out; but attention seems never to have been called to some essential particulars in which it differs from all other forms of nystagmus.

Ordinary horizontal nystagmus is, of course, conjugate in its character, the antero-posterior axes of the two eyes remaining parallel to one another all the time. In this form, however, when the movements are bilateral and horizontal, the eyes incline alternately towards and away from one another; in other words, it is convergent nystagmus. In most of the cases—perhaps in as many as two-thirds—the shaking of the eyeballs is so small in extent and so rapid that the unaided eye cannot determine the type. In those, however, in which the movements are wider and slower than usual their convergent character can be made out with ease and certainty.

The writer has for years been aware of this peculiarity, but has only recently had its unique nature and importance pointed out to him by Dr. W. G. Sym. The existence of a convergent form of nystagmus is not generally recognized, even in large and recent works of ophthalmology, nor is any reference to it to be found in books on the diseases of children.

The association of this peculiar type of nystagmus with head-shaking is not without interest in connection with the etiology of that condition. Although, as has been pointed out elsewhere,[1] rickets, deficient daylight and general weak health are important factors in its causation, spasmus nutans in infants is, at bottom, a co-ordination-neurosis. It develops during the months in which the infant is slowly learning to co-ordinate the movements of his eyes with those of his head,

[1] On the Etiology of Head-Shaking with Nystagmus (Spasmus Nutans) in Infants, Dr. A. Jacobi's *Festschrift*, May, 1900; and *Scot. Med. and Surg. Journ*, July, 1900.

and it affects the muscles which have to do with these movements. Were the nystagmus which is present of the conjugate type it would not be in keeping with the other symptoms, because the conjugate movements of the eyes are of a different order altogether from those of the head-shaking. They are not movements acquired in infancy, but are in full play when the child is born; and they are not purposive in character. Those, however, concerned in the convergent type are distinctly purposive, and are gradually acquired and perfected by practice during infancy.

In those cases of spasmus nutans in which the nystagmus is rotatory, there is likewise a peculiarity in the extent and character of the movements. In the ordinary rotatory nystagmus seen under other circumstances, the movement consists of a simple rotation of the globe round its antero-posterior axis, the central point of the cornea remaining practically unaltered in position. In the rotatory nystagmus of head-shaking, however, the eye movements are more of the nature of circumduction than pure rotation. In them the central point of the cornea passes through an ellipse or some other more or less irregular rounded figure.

In conclusion, it may be pointed out that there are some other ways in which the nystagmus of spasmus nutans differs from the ordinary form. For example, it is often unilateral, instead of being very rarely so; and it is often vertical or rotatory, instead of being nearly always horizontal. The direction of the movements, also, is occasionally different in the two eyes—that is, vertical or rotatory in one eye, and horizontal (at the same time) in the other, this state of matters being never seen in ordinary nystagmus. Lastly, it is invariably recovered from within a certain number of months.

CAN INTERSTITIAL KERATITIS BE PREVENTED IN THE OFFSPRING OF SYPHILITICS?

Peter A. Callan (*American Gynecological and Obstetrical Journal*, February, 1901) assumes that the majority of cases are due to hereditary syphilis, and the question may arise as to whether it can be avoided. The authors conclusions are: "The offspring of syphilitics show the evidences of the inherited taint in only a slight percentage of the cases; that is,

if we except those children under five years of age. Only a slight percentage of such children develop interstitial keratitis. When the disease attacks one eye, all our treatment appears to be powerless to prevent its development in the fellow eye. We may possibly delay, but we can not prevent, the outbreak of interstitial keratitis in certain cases."

NOTES ON THE BACTERIOLOGY OF THE CONJUNCTIVAL SAC AND ITS BEARING ON SURGICAL PROCEDURE.

P. Chalmers Jackson (*Annals of Ophthalmology*, January, 1901) examined the secretion from fifty healthy conjunctivæ, thirteen of which were found to be sterile. In the remainder he found the following bacteria: Staphylococcus pyogenes aureus, staphylococcus pyogenes albus, staphylococcus epidermidis albus, aerobacillus citreus, xerosis bacillus, bacillus coli communis, and bacillus subtilis.

The author's conclusions are as follows:

1. That the pyogenic or pus-producing organisms are found in the normal conjunctival secretion, although probably in attenuated form.

2. That under normal conditions they do not propagate.

3. That the eye under normal conditions is bountifully supplied with means of antagonizing bacterial growth.

4. That diminished resistance, such as occurs in inflammations of the membrane in operative interference, alters the nutritive nature of the secretion and probably converts it into a more suitable medium for germ life.

5. That the secretion of the eye is not an antiseptic in itself.

6. That strong antiseptics in the conjunctival sac diminish the resistance and place the eye on a lower plane to resist germ invasion.

7. That much attention should be given to washing out residual bacteria prior to operation.

8. That as much care should be taken in regard to antiseptics and cleanliness in the external preparation of both patient and operator as is adopted by the general surgeon of modern times, as, while the danger of suppuration is more remote, the result if it occurs is more disastrous.